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N-acetyl-cysteine may prevent COVID-19-associated cytokine storm and acute respiratory distress syndrome



To the Editor.

Accumulating evidence suggests that a subgroup of patients with severe COVID-19 might have a cytokine storm syndrome associated with acute respiratory distress syndrome (ARDS), multiple organ failure and increased mortality. This syndrome is characterised by increased interleukin (IL)-2, IL-7, granulocyte colony stimulating factor, interferon-y inducible protein 10, monocyte chemoattractant protein 1, macrophage inflammatory protein 1-α, and tumour necrosis factor

N-Acetylcysteine (NAC), a well-known mucolytic agent used in respiratory infections, is a thiol-containing free-radical scavenger and a precursor of glutathione [2]. Reactive oxygen species and oxidative stress activate important redox-sensitive transcription factors like NFκB and activator protein-1, which lead to the co-ordinate expression of proinflammatory genes of IL-6, IL-8, and TNF- α [3].

The beneficial action of 1200 mg/d of oral NAC in respiratory diseases has been previously demonstrated in prevention of chronic obstructive pulmonary disease exacerbations [2]. Moreover, a recent study including patients with community-acquired pneumonia, showed that the addition of this dose of NAC to conventional treatment improves oxidative stress and inflammatory response [4]. The positive effects of NAC in viral lower respiratory tract infections have been associated with inhibition of IL-8, IL-6, and TNF-α expression and release in alveolar type II cells infected with influenza virus A and B and respiratory syncytial virus [5].

The results of these studies offer reasonable basis for the addition of 1200 mg/d oral NAC on therapeutic schemes of patients with COVID-19, as a measure that could potentially prevent the development of the cytokine storm syndrome and ARDS. This hypothesis is worth clarifying in appropriately designed clinical studies.

Prior presentation

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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